frequently be avoided if the subjects show the fixity of purpose described here; their determination to minimise the wind-chill factor (by digging a substantial snow cave) and to overcome the urge to sleep undoubtedly prevented a serious outcome for this party, which carried no specific equipment to spend a night out near the summit of Everest.

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Heart block and autoimmunity

SIR,—In reply to Dr F I Lee (29 November, p 524) the four patients quoted in our paper who had chronic heart block and hypothyroidism did not develop these disorders simultaneously. In three patients heart block occurred between four and eight years after they had become clinically hypothyroid, while they were on replacement therapy. The fourth patient, whose history was quoted, was clinically euthyroid when heart block developed, but a raised thyroid stimulating hormone level at this time, together with evidence of multiple autoimmune disorders, led to the institution of thyroid replacement therapy, which has not, after 12 months, reversed his conduction disturbance. None of these patients had evidence of ischaemic heart disease but all had thyroid autoantibodies present. As in Dr Lee's case, the irreversibility of heart block by administered thyroid hormone suggests that a direct metabolic effect alone is insufficient to explain these data.

Permanent damage to the sinoatrial node has been reported in thyrotoxicosis,[1] and recently "lone" atrial fibrillation has been associated both with "silent" thyrotoxicosis and, surprisingly, with early hypothyroidism.[2] An autoimmune aetiology seems to be a possible unifying factor to explain these observations, and it is possible that circulating immune complexes, which have been proposed as a cause of the skeletal muscle damage in thyrotoxic ocular myositis,[3] could be one of the mechanisms by which the heart is damaged in these autoimmune disorders.

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First-aid treatment of poisoning

SIR,—Your issue of the 29 November contained two items on this topic, a letter on "Salt overdosage" (p 517) and the leading article on "Childhood poisoning: prevention and first-aid management" (p 483). On the first item we informed you in 1974 (9 November, p 342) that the joint manual of the voluntary aid societies would be modified to omit the use by first-aiders of the induction of emesis in the treatment of swallowed poisons; this has now been done.

At the time we decided to make this modification we considered recommending the use of ipecacuanha in the first-aid treatment of childhood poisoning. We concluded that to try to define for first-aiders the indications for this procedure would lead to confusion and that the occasions on which first-aiders would be required to administer ipecacuanha were so few that it would be wasteful of resources and contrary to the aim of simplicity in first-aid equipment to provide it.

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Chronic peritoneal dialysis for diabetic nephropathy

SIR,—Your leading article (16 August, p 397) entitled "Renal transplantation in diabetes," concludes that renal transplantation is probably the most promising treatment for patients with end-stage diabetic kidney disease. Renal transplantation may be the preferred treatment in younger diabetic patients. The mean age of those transplanted in Minneapolis was 34 years, the oldest patient being 49.[1] Success is more likely in those lacking severe cardiovascular disease and other complications of diabetes and in circumstances where a living donor is available. However, such patients make up only a small fraction of the total number of patients with end-stage uraemia due to diabetic nephropathy.

In the United States, with the recent enactment of new social security Medicare laws, renal replacement therapy is becoming feasible for nearly all patients with end-stage renal disease. The magnitude of the problem is great. It was conservatively estimated that in the US approximately 15 diabetic patients develop end-stage nephropathy per million population per year (3200/year in the US).[2] Many of these patients are in the older age group and have multiple complications of diabetes. For this group of patients transplantation is not feasible. The difficult medical and moral problem has been whether these patients are best treated by dialysis or by "conservative measures," which implies that they will soon die as the consequence of the disease. The results of dialysis therapy in this group often prove less satisfactory than in non-diabetic uraemic patients, but an increasing number of dialysis centres are accepting these patients for care. Although the results of haemodialysis in this population have for the most part not been good, maintenance peritoneal dialysis may offer a promising alternative in the patient with diabetes and end-stage uraemia.[3,4] The mean age of the eight diabetic patients we have treated with maintenance peritoneal dialysis at this centre is 54 years. This mode of therapy may be better suited for the patient with severe cardiovascular disease who is prone to haemodynamic instability and arrhythmias. Since rapid fluctuations of blood pressure are less frequent and systemic anticoagulation is not employed eye disease may not progress at as rapid a rate with haemodialysis, while problems of vascular access necessary for haemodialysis are obviated.

None of the current methods available for managing end-stage uraemia in diabetic patients provide the chances for rehabilitation seen in the uraemic patient without diabetes. In the older diabetic patient with severe cardiovascular disease it is likely that the physician, the patient, and the patient's family must accept more limited goals. Under such circumstances peritoneal dialysis may offer certain advantages over haemodialysis: it can be safely carried out at home even without the presence of another person, and a patient can be trained for self-care at home in less time than is required for haemodialysis. Thus a well-motivated patient may achieve satisfactory rehabilitation while peritoneal dialysis is performed during sleep three to five nights per week.

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Immunisation against whooping cough

SIR,—During the past year you have published several reports and letters about whooping cough, including notably the report of the Joint Committee on Vaccination and Immunisation (20 September, p 687). None of these publications offered data for epidemiological analysis, so readers were dependent for guidance upon the recommendations of the committee and of your leading article (25 October, p 186). In this article you referred to "evidence" that current vaccines give such a high degree of protection that experts are "almost unanimous" in recommending its use routinely, thus endorsing the view of the committee that the decline in the incidence of whooping cough since 1958 is attributable to immunisation.

Immediately after reading this leading article I sent to you a short paper, based largely upon data available to the committee, in which I questioned the assumption that routine immunisation was responsible for the decline in the incidence of whooping cough. I drew attention also to the continuing importance of overcrowding and other socioeconomic factors which appeared, in the UK generally and in Glasgow especially, to be at least as important as lack of immunisation in determining the distribution of recent outbreaks. So that the "evidence" could be assessed independently I stated that the raw data, national and local, on which my conclusions were based would be made available to anyone who wanted it through the Communicable Diseases (Scotland) Unit at Ruchill Hospital, Glasgow G20.

Since you have chosen to reject this paper even after amendment and since you cannot publish my raw data I am wondering if you will extend to me the courtesy of your columns to ask for the "evidence" on which the committee's report and your leading article are based. Meanwhile, for anyone who wants it, my data are on file in the Communicable Diseases (Scotland) Unit at Ruchill Hospital. Our article will be submitted for information and publication in other quarters.

There is a further point of immediate importance. Because of continuing doubts and lack of "evidence" about the safety and efficacy of immunisation against whooping cough, immunisation of children is declining sharply. The decline is most marked in districts where, according to our "evidence," the risk of contracting infection is highest. Unfortunately, in parallel with diminishing use of pertussis vaccine there is a fall in other forms of immunisation in childhood, possibly because unanswered questions about pertussis vaccine are discouraging routine immunisations against diphtheria, poliomyelitis, and tetanus. For this reason also I trust that the "evidence" on which you clearly rely will not be unduly delayed in finding its way into your columns.

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The problem of rosacea

SIR,—It is true that, as reported in your leading article (15 November, p 366) I "took on the task of critically re-examining the main ideas in the light of [my] experience of 92 patients with rosacea; none could be supported." However, while agreeing that "there are still vacancies in the cowshed," I would point out that you made no reference to a suggestion that I have made previously as to a potential new occupant.

We have pointed out[1] that the upper dermis in rosacea is quite abnormal and shows evidence of both solar elastotic degeneration considerably in advance of what might reasonably be expected for a group of middle-aged Britishers and other dystrophic changes that are not easily categorised. Autoradiographs after injection of tritiated thymidine and enzyme histochemical tests have suggested[2] that small dermal blood vessels are also involved in rosacea (probably secondarily). It is my belief, based on these findings, that the primary disorder is a dermal dystrophy resulting from "weathering" (sun, wind, and cold) and an inherent susceptibility to this process. The dermal attenuation produced in this way causes lack of dermal support for the subpapillary venous plexus, allowing these channels (and neighbouring lymphatics) to dilate enormously. The flushing seen in rosacea is in all probability the result of the vessel dilatation—not its cause. The dilated vessels could become incompetent in addition as a result of the persistent and extreme pooling seen in them and this in turn may lead to diffusion of injurious macromolecules and mediators of the inflammatory process into the dermis. This hypothetical process may be summarised in the diagram.

I would not wish the above suggested "occupant of the cowshed" to become a sacred cow. However, there is sufficient evidence for this hypothesis to be further tested and I hope that this letter will stimulate the cow's dissection rather than its perpetual milking.

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Plasma free fatty acids and ventricular fibrillation

SIR,—Dr C A Sykes and his colleagues (27 December, p 735) report the relationship between plasma free fatty acid (FFA) concentrations in 23 diabetic and 24 non-diabetic patients with myocardial infarction and the incidence of ventricular fibrillation (VF). They showed that the plasma FFA levels were similar in diabetics who developed VF and in those who did not. But they are wrong in reaching the same conclusion for non-diabetics, as the following reassembly of their own data indicates.

Patients	Mean plasma FFA (mmol l) ± 1 SD	Significance of difference
Non-diabetics with VF (5)	1·76 ± 0·61	P<0·05
Non-diabetics without VF (19)	1·24 ± 0·40	
Diabetics with VF (5)	1·30 ± 0·59	Not significant
Diabetics without VF (18)	1·46 ± 0·62	

The highest mean plasma FFA is in the subgroup of non-diabetics who developed VF and the concentrations considerably exceed the "threshold level" of about 1·2 mmol/l, which approximates to a 2:1 molar binding ratio with albumin. Higher concentrations are associated with less firmly bound FFA and, as we showed[1] and originally proposed,[2] with a greater incidence of serious ventricular arrhythmias. Therefore I regard the findings of Dr Sykes and his colleagues as contradicting one of their own conclusions and, at the same time, as additional supportive evidence to the other studies[3-5] which have shown a positive correlation in non-diabetic patients with myocardial infarction between elevated plasma FFA or glycerol levels and ventricular arrhythmias.

The authors appear to be justified, however, in reaching their other conclusion that plasma FFA levels do not correlate with VF in diabetic patients. Why should this be? One explanation might be that the mean plasma FFA concentrations were not very greatly elevated in these particular patients at a mean level of 1·30 mmol/l. Another might be that the ischaemic myocardium is partly protected from the adverse effect of high plasma FFA because of the relatively high intracellular glucose concentrations likely to be present, particularly in those receiving insulin. The same authors[6] have previously shown that VF is less common in insulin-treated diabetics compared with these on oral drugs but, unfortunately, it is not possible to determine from the recent report whether the five diabetics who developed VF were on insulin or oral drugs.

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Dietary fibre: search for the facts

SIR,—A letter from Mr C L Copeland, executive director of the Flour Advisory Bureau (15 November, p 404), was headed "Dietary fibre: search for the facts." Perhaps, as the reporter/producer concerned with the "recent BBC TV programme concerning food," I may be allowed to correct his facts.

Mr Copeland said that "once again, in a recent BBC TV programme concerning food, a commentator has had no hesitation in linking a list of serious diseases, including cancer of the colon, 'with all that lost fibre,' singling out white bread critically and claiming that some consumers *have* to obtain their fibre from a certain (named) proprietary pharmaceutical product." I am sure your readers will be able to make up their minds as to the correctness of Mr Copeland's synopsis if I quote what I did say.

The context of the programme was a comparison between 1875 (the date of the first effective food and drugs Act) and 1975. I said "compared to a century ago, the amount of roughage we get from bread has dropped dramatically, down to only one-sixth. That's the roughage that comes from the fibre of the wheat grain." Then came a sequence illustrating the baking of traditional white bread and comparing its water content with that of a modern plant-baked loaf.

I then said: "and what about all that lost fibre? There is a growing medical theory—